L Number	Hits	Search Text	DB	Time stamp
1	19	palli-subba-reddy.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/04/22 15:30
2	5	kapitskaya-marianna-zinovjevna.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/04/22 15:31
3	12	cress-dean-ervin.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/04/22 15:31
4	27	two same hybrid same system same ecdysone	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/04/22 15:32

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NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
NEWS	2		"Ask CAS" for self-help around the clock
NEWS	3	JAN 27	Source of Registration (SR) information in REGISTRY updated and searchable
NEWS	4	JAN 27	A new search aid, the Company Name Thesaurus, available in CA/CAplus
NEWS	5	FEB 05	German (DE) application and patent publication number format changes
NEWS	6	MAR 03	MEDLINE and LMEDLINE reloaded
NEWS	7	MAR 03	MEDLINE file segment of TOXCENTER reloaded
NEWS	8	MAR 03	FRANCEPAT now available on STN
NEWS	9	MAR 29	Pharmaceutical Substances (PS) now available on STN
NEWS	10	MAR 29	WPIFV now available on STN
NEWS	11	MAR 29	No connect hour charges in WPIFV until May 1, 2004
NEWS	12	MAR 29	New monthly current-awareness alert (SDI) frequency in RAPRA

NEWS EXPRESS	MARCH 31 CURRENT WINDOWS VERSION IS V7.00A, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 13 APRIL 2004
NEWS HOURS	STN Operating Hours Plus Help Desk Availability
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FILE 'MEDLINE' ENTERED AT 16:01:26 ON 22 APR 2004

FILE 'BIOSIS' ENTERED AT 16:01:26 ON 22 APR 2004

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FILE 'CAPLUS' ENTERED AT 16:01:26 ON 22 APR 2004
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L2 5 KAPITSKAYA MARIANNA ZINOVJEVNA

=> s cress dean ervin /au
L3 11 CRESS DEAN ERVIN

=> s ecdysone (s) ligand (s) bind? (s) domain (s) transactiva? (s) two (s) hybrid
L4 1 ECDYSONE (S) LIGAND (S) BIND? (S) DOMAIN (S) TRANSACTIVA? (S) TWO (S) HYBRID

=> d l4 ibib kwic

L4 ANSWER 1 OF 1 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2003139225 EMBASE
TITLE: Improved ecdysone receptor-based inducible gene regulation system.
AUTHOR: Palli S.R.; Kapitskaya M.Z.; Kumar M.B.; Cress D.E.
CORPORATE SOURCE: S.R. Palli, Department of Entomology, College of Agriculture, University of Kentucky, Lexington, KY 40546, United States. RPALLI@UKY.EDU
SOURCE: European Journal of Biochemistry, (2003) 270/6 (1308-1315). Refs: 37
ISSN: 0014-2956 CODEN: EJBCAI
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
LANGUAGE: English
SUMMARY LANGUAGE: English
AB To develop an **ecdysone** receptor (EcR)-based inducible gene regulation system, several constructs were prepared by fusing DEF **domains** of Choristoneura fumiferana EcR (CfEcR), C. fumiferana ultraspiracle (CfUSP), Mus musculus retinoid X receptor (MmRXR) to either GAL4 DNA **binding domain** (DBD) or VP16 activation **domain**. These constructs were tested in mammalian cells to evaluate their ability to **transactivate** luciferase gene placed under the control of GAL4 response elements and synthetic TATAA promoter. A **two-hybrid** format switch, where GAL4 DBD was fused to CfEcR (DEF) and VP16 AD was fused to MmRXR (EF) was found. . . to be the best combination. It had the lowest background levels of reporter gene activity in the absence of a **ligand** and the highest level of reporter gene activity in the presence of a **ligand**. Both induction and turn-off responses were fast. A 16-fold induction was observed within 3 h of **ligand** addition and increased to 8942-fold by 48 h after the addition of **ligand**. Withdrawal of the **ligand** resulted in 50% and 80% reduction in reporter gene activity by 12 h and 24 h, respectively.

=> d his

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     FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS' ENTERED AT 16:01:26 ON 22 APR 2004
L1            45 S PALLI SUBBA REDDY /AU
L2             5 S KAPITSKAYA MARIANNA ZINOVJEVNA /AU
L3            11 S CRESS DEAN ERVIN /AU
L4             1 S ECDYSONE (S) LIGAND (S) BIND? (S) DOMAIN (S) TRANSACTIVA? (S)

=> s ligand (s) bind? (s) domain (s) transactiva? (s) two (s) hybrid (s) retinoid
L5            15 LIGAND (S) BIND? (S) DOMAIN (S) TRANSACTIVA? (S) TWO (S) HYBRID
                 (S) RETINOID

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6            15 DUP REM L5 (0 DUPLICATES REMOVED)

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L6   ANSWER 1 OF 15  EMBASE  COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
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ACCESSION NUMBER:    2003345065  EMBASE
TITLE:               The cell death regulator GRIM-19 is an inhibitor of signal
                     transducer and activator of transcription 3.
AUTHOR:              Zhang J.; Yang J.; Roy S.K.; Tininini S.; Hu J.; Bromberg
                     J.F.; Poli V.; Stark G.R.; Kalvakolanu D.V.
CORPORATE SOURCE:    D.V. Kalvakolanu, Greenebaum Cancer Center, Dept. of
                     Microbiology and Immunology, Univ. of Maryland School of
                     Medicine, Baltimore, MD 21201, United States.
                     dkalvako@umaryland.edu
SOURCE:              Proceedings of the National Academy of Sciences of the
                     United States of America, (5 Aug 2003) 100/16 (9342-9347).
                     Refs: 50
                     ISSN: 0027-8424  CODEN: PNASA6
COUNTRY:             United States
DOCUMENT TYPE:       Journal; Article
FILE SEGMENT:        016     Cancer
                     029     Clinical Biochemistry
                     037     Drug Literature Index
LANGUAGE:            English
SUMMARY LANGUAGE:    English
AB   GRIM-19 (gene associated with retinoid-IFN-induced mortality
     19), isolated as a cell death activator in a genetic screen used to define
     mechanisms involved in IFN-β- and.  .  .  a growth advantage to
     cells. To understand the molecular bases for its cell death regulatory
     activity, we used a yeast two-hybrid screen and
     identified that the transcription factor STAT3 (signal transducer and
     activator of transcription 3) binds to GRIM-19. GRIM-19 inhibits
     transcription driven by activation of STAT3, but not STAT1. It neither
     inhibits the ligand-induced activation of STAT3 nor blocks its
     ability to bind to DNA. Mutational analysis indicates that the
     transactivation domain of STAT3, especially residue
     S727, is required for GRIM-19 binding. Because GRIM-19 does not
     bind significantly to other STATs, our studies identify a specific
     inhibitor of STAT3. Because constitutively active STAT3 up-regulates
     antiapoptotic genes to.  .  .

L6   ANSWER 2 OF 15  EMBASE  COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
     on STN
ACCESSION NUMBER:    2003139225  EMBASE
TITLE:               Improved ecdysone receptor-based inducible gene regulation
                     system.
AUTHOR:              Palli S.R.; Kapitskaya M.Z.; Kumar M.B.; Cress D.E.
CORPORATE SOURCE:    S.R. Palli, Department of Entomology, College of
                     Agriculture, University of Kentucky, Lexington, KY 40546,
                     United States. RPALLI@UKY.EDU
SOURCE:              European Journal of Biochemistry, (2003) 270/6 (1308-1315).
```

Refs: 37
ISSN: 0014-2956 CODEN: EJBCAI
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
LANGUAGE: English
SUMMARY LANGUAGE: English
AB To develop an ecdysone receptor (EcR)-based inducible gene regulation system, several constructs were prepared by fusing DEF **domains** of Choristoneura fumiferana EcR (CfEcR), C. fumiferana ultraspiracle (CfUSP), Mus musculus **retinoid** X receptor (MmRXR) to either GAL4 DNA **binding domain** (DBD) or VP16 activation **domain**. These constructs were tested in mammalian cells to evaluate their ability to **transactivate** luciferase gene placed under the control of GAL4 response elements and synthetic TATAA promoter. A **two-hybrid** format switch, where GAL4 DBD was fused to CfEcR (DEF) and VP16 AD was fused to MmRXR (EF) was found. . . to be the best combination. It had the lowest background levels of reporter gene activity in the absence of a **ligand** and the highest level of reporter gene activity in the presence of a **ligand**. Both induction and turn-off responses were fast. A 16-fold induction was observed within 3 h of **ligand** addition and increased to 8942-fold by 48 h after the addition of **ligand**. Withdrawal of the **ligand** resulted in 50% and 80% reduction in reporter gene activity by 12 h and 24 h, respectively.

L6 ANSWER 3 OF 15 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2002309355 EMBASE
TITLE: Requirement of helix 1 and the AF-2 domain of the thyroid hormone receptor for coactivation by PGC-1.
AUTHOR: Wu Y.; Delerive P.; Chin W.W.; Burris T.P.
CORPORATE SOURCE: T.P. Burris, Gene Regulation, Lilly Research Laboratories, Lilly Corporate Center, Indianapolis, IN 46285, United States. Burris_Thomas_P@lilly.com
SOURCE: Journal of Biological Chemistry, (15 Mar 2002) 277/11 (8898-8905).
Refs: 56
ISSN: 0021-9258 CODEN: JBCHA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Although PGC-1 (peroxisome proliferator-activated receptor-γ coactivator-1) has been previously shown to enhance thyroid hormone receptor (TR)/**retinoid** X receptor-mediated ucp-1 gene expression in a **ligand**-induced manner in rat fibroblast cells, the precise mechanism of PGC-1 modulation of TR function has yet to be determined. In this study, we show that PGC-1 can potentiate TR-mediated **transactivation** of reporter genes driven by natural thyroid hormone response elements both in a **ligand**-dependent and **ligand**-independent manner and that the extent of coactivation is a function of the thyroid hormone response element examined. Our data also show that PGC-1 stimulation of TR activity in terms of Gal4 DNA-**binding domain** fusion is strictly **ligand**-dependent. In addition, an E457A AF-2 mutation had no effect on the **ligand**-induced PGC-1 enhancement of TR activity, indicating that the conserved charged residue in AF-2 is not essential for this PGC-1 function. Furthermore, GST pull-down and mammalian **two-hybrid** assays demonstrated that the PGC-1 LXXLL motif is required for **ligand**-induced PGC-1/TR interaction. This agonist-dependent PGC-1/TR interaction also requires both helix 1 and the AF-2 region of the TR **ligand-binding domain**. Taken together,

these results support the notion that PGC-1 is a bona fide TR coactivator and that PGC-1 modulates TR. . .

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ACCESSION NUMBER: 2002129281 EMBASE
TITLE: Nuclear receptor corepressor-dependent repression of peroxisome-proliferator-activated receptor δ-mediated transactivation.
AUTHOR: Krogsdam A.-M.; Nielsen C.A.F.; Neve S.; Holst D.; Helledie T.; Thomsen B.; Bendixen C.; Mandrup S.; Kristiansen K.
CORPORATE SOURCE: K. Kristiansen, Department of Biochemistry, University of Southern Denmark, Odense University, Campusvej 55, DK-5230 Odense M, Denmark. kak@bmb.sdu.dk
SOURCE: Biochemical Journal, (1 Apr 2002) 363/1 (157-165).
Refs: 52
ISSN: 0264-6021 CODEN: BIJOAK
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The nuclear receptor corepressor (NCoR) was isolated as a peroxisome-proliferator-activated receptor (PPAR) δ interacting protein using the yeast **two-hybrid** system. NCoR interacted strongly with the **ligand-binding domain** of PPARδ, whereas interactions with the **ligand-binding domains** of PPARγ and PPARα were significantly weaker. PPAR-NCoR interactions were antagonized by **ligands** in the **two-hybrid** system, but were **ligand**-insensitive in in vitro pull-down assays. Interaction between PPARδ and NCoR was unaffected by coexpression of **retinoid** X receptor (RXR) α. The PPARδ-RXRα heterodimer bound to an acyl-CoA oxidase (ACO)-type peroxisome-proliferator response element recruited a glutathione S-transferase-NCoR fusion protein in a **ligand**-independent manner. Contrasting with most other nuclear receptors, PPARδ was found to interact equally well with interaction **domains** I and II of NCoR. In transient transfection experiments, NCoR and the related silencing mediator for **retinoid** and thyroid hormone receptor (SMRT) were shown to exert a marked dose-dependent repression of **ligand**-induced PPARδ-mediated **transactivation**; in addition, **transactivation** induced by the cAMP-elevating agent forskolin was efficiently reduced to basal levels by NCoR as well as SMRT coexpression. Our results suggest that the **transactivation** potential of liganded PPARδ can be fine-tuned by interaction with NCoR and SMRT in a manner determined by the expression. . .

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ACCESSION NUMBER: 2000063976 EMBASE
TITLE: Activation of orphan receptor-mediated transcription by Ca2+/calmodulin-dependent protein kinase IV.
AUTHOR: Kane C.D.; Means A.R.
CORPORATE SOURCE: A.R. Means, Dept. Pharmacology Cancer Biology, Duke University, Medical Center, PO Box 3813, Durham, NC 27710, United States. means001@mc.duke.edu
SOURCE: EMBO Journal, (15 Feb 2000) 19/4 (691-701).
Refs: 55
ISSN: 0261-4189 CODEN: EMJODG
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English

SUMMARY LANGUAGE: English

AB **Retinoid**-related receptor α (RORα) is an orphan nuclear receptor that constitutively activates transcription from its cognate response element. We show that. . . selective and does not occur with either the thyroid hormone or estrogen receptor. CaMKIV does not phosphorylate RORα or its **ligand-binding domain** (LBD) in vitro, although the LED is essential for **transactivation**. Therefore, the RORα LED was used in the mammalian **two-hybrid** assay to identify a single class of small peptide molecules containing LXXLL motifs that interacted with greater affinity in the. . .

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ACCESSION NUMBER: 1999416820 EMBASE

TITLE: A nuclear factor, ASC-2, as a cancer-amplified transcriptional coactivator essential for ligand-dependent transactivation by nuclear receptors in vivo.

AUTHOR: Lee S.-K.; Anzick S.L.; Choi J.-E.; Bubendorf L.; Guan X.-Y.; Jung Y.- K.; Kallioniemi O.P.; Kononen J.; Trent J.M.; Azorsa D.; Jhun B.-H.; Jae Hun Cheong; Young Chul Lee; Meltzer P.S.; Jae Woon Lee

CORPORATE SOURCE: J.W. Lee, Center for Ligand and Transcription, Hormone Research Center, Chonnam National University, Kwangju 500-757, Korea, Republic of. jlee@chonnam.chonnam.ac.kr

SOURCE: Journal of Biological Chemistry, (26 Nov 1999) 274/48 (34283-34293).
Refs: 76
ISSN: 0021-9258 CODEN: JBCHA3

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 005 General Pathology and Pathological Anatomy
016 Cancer
029 Clinical Biochemistry

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Many transcription coactivators interact with nuclear receptors in a **ligand**- and C-terminal **transactivation** function (AF2)-dependent manner. We isolated a nuclear factor (designated ASC-2) with such properties by using the **ligand-binding domain** of **retinoid** X receptor as a bait in a yeast **two- hybrid** screening. ASC-2 also interacted with other nuclear receptors, including retinoic acid receptor, thyroid hormone receptor, estrogen receptor α, and glucocorticoid. . . and transcription integrators CBP/p300 and SRC-1. In transient cotransfections, ASC-2, either alone or in conjunction with CBP/p300 and SRC-1, stimulated **ligand**-dependent **transactivation** by wild type nuclear receptors but not mutant receptors lacking the AF2 **domain**. Consistent with an idea that ASC-2 is essential for the nuclear receptor function in vivo, microinjection of anti-ASC-2 antibody abrogated the **ligand**-dependent **transactivation** of retinoic acid receptor, and this repression was fully relieved by coinjection of ASC-2-expression vector. Surprisingly, ASC-2 was identical to. . .

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ACCESSION NUMBER: 1999173046 EMBASE

TITLE: The autonomous transactivation domain in helix H3 of the vitamin D receptor is required for transactivation and coactivator interaction.

AUTHOR: Kraichely D.M.; Collins III J.J.; DeLisle R.K.; MacDonald P.N.

CORPORATE SOURCE: P.N. MacDonald, St. Louis Univ. School of Medicine,

Pharmacolog./Physiolog. Sci. Dept., 1402 South Grand Blvd., St. Louis, MO 63104, United States. macdonal@slu.edu
SOURCE: Journal of Biological Chemistry, (14 May 1999) 274/20 (14352-14358).
Refs: 39
ISSN: 0021-9258 CODEN: JBCHA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
AB A **ligand**-inducible **transactivation** function (AF-2) exists in the extreme carboxyl terminus of the vitamin D receptor (VDR) that is essential for 1α,25-dihydroxyvitamin D3 (1,25-(OH)2D3)-activated transcription and p160 coactivator interaction. Crystallographic data of related nuclear receptors suggest that **binding** of 1,25-(OH)2D3 by VDR induces conformational changes in the **ligand -binding domain** (LBD), the most striking of which is a packing of the AF-2 helix onto the LBD adjacent to helices H3. . . . this study, a panel of VDR helix H3 mutants was generated, and residues in helix H3 that are important for **ligand**-activated transcription by the full-length VDR were identified. In particular, one mutant (VDR (Y236A)) exhibited normal **ligand binding** and heterodimerization with the **retinoid** X receptor (RXR) but was transcriptionally inactive. Yeast **two-hybrid** studies and in vitro protein interaction assays demonstrated that VDR (Y236A) was selectively impaired in interaction with AF-2-interacting coactivator proteins. . . . in the mechanism of VDR-mediated transcription, and they support the concept that helix H3 functions in concert with the AF-2 **domain** to form a **transactivation** surface for **binding** the p160 class of nuclear receptor coactivators.

L6 ANSWER 8 OF 15 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 1999328256 EMBASE
TITLE: NRIF3 is a novel coactivator mediating functional specificity of nuclear hormone receptors.
AUTHOR: Li D.; Desai-Yajnik V.; Lo E.; Schapira M.; Abagyan R.; Samuels H.H.
CORPORATE SOURCE: H.H. Samuels, Division of Molecular Endocrinology, Department of Medicine, New York Univ. School of Medicine, 550 First Ave., New York, NY 10016, United States. samueh01@mcrcr.med.nyu.edu
SOURCE: Molecular and Cellular Biology, (1999) 19/10 (7191-7202).
Refs: 87
ISSN: 0270-7306 CODEN: MCEBD4
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
AB . . . (designated NRIF3) that exhibits a distinct receptor specificity. Fluorescence microscopy shows that NRIF3 localizes to the cell nucleus. The yeast **two-hybrid** and/or in vitro **binding** assays indicated that NRIF3 specifically interacts with the thyroid hormone receptor (TR) and **retinoid** X receptor (RXR) in a **ligand**- dependent fashion but does not **bind** to the retinoic acid receptor, vitamin D receptor, progesterone receptor, glucocorticoid receptor, or estrogen receptor. Functional experiments showed that NRIF3 significantly potentiates TR- and RXR-mediated **transactivation** in vivo but has little effect on other examined nuclear receptors. **Domain** and mutagenesis analyses indicated that a novel C-terminal **domain** in NRIF3 plays an essential role in its specific interaction with liganded TR and RXR while the N-terminal

LXXLL motif plays a minor role in allowing optimum interaction. Computer modeling and subsequent experimental analysis suggested that the C-terminal **domain** of NRIF3 directly mediates interaction with liganded receptors through an LXXIL (a variant of the canonical LXXLL) module while the. . .

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ACCESSION NUMBER: 2000303761 EMBASE
TITLE: Estrogen receptor, a common interaction partner for a subset of nuclear receptors.
AUTHOR: Lee S.-K.; Choi H.-S.; Song M.-R.; Lee M.-O.; Lee J.W.
CORPORATE SOURCE: Dr. J.W. Lee, College of Pharmacy, Hormone Research Center, Chonnam National University, Kwangju 500-757, Korea, Republic of. jlee@chonnam.chonnam.ac.kr
SOURCE: Molecular Endocrinology, (1998) 12/8 (1184-1192).
Refs: 59
ISSN: 0888-8809 CODEN: MOENEN
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Nuclear receptors regulate transcription by **binding** to specific DNA response elements as homodimers or heterodimers. Herein, the yeast and mammalian **two-hybrid** tests as well as glutathione-S-transferase pull-down assays were exploited to demonstrate that estrogen receptor (ER) directly **binds** to a subset of nuclear receptors through protein-protein interactions between **ligand-binding domains**. These receptors include hepatocyte nuclear factor 4, thyroid hormone receptor (TR), retinoic acid receptor (RAR), ERβ, and **retinoid** X receptor (RXR). In yeast cells, a LexA fusion protein to the human ER **ligand-binding domain** (LexA/ER-LBD) was an inert **transactivator** of a LacZ reporter gene controlled by upstream LexA-**binding** sites. However, LexA/ER-LBD differentially modulated the LacZ reporter gene expression when coexpressed with native TRs, RARs, or RXRs. Similarly, cotransfection of these receptors in CV1 cells up- or down-regulated **transactivations** by ER. From these results, we propose that ER is a common interaction partner for a subset of receptors, and. . .

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ACCESSION NUMBER: 1999005483 EMBASE
TITLE: Differential modulation of transcriptional activity of oestrogen receptors by direct protein-protein interactions with retinoid receptors.
AUTHOR: Song M.-R.; Lee S.-K.; Seo Y.-W.; Choi H.-S.; Lee J.W.; Lee M.-O.
CORPORATE SOURCE: M.-O. Lee, Department of Microbiology, Yonsei University College Medicine, Seoul 120-752, Korea, Republic of. molee@yumc.yonsei.ac.kr
SOURCE: Biochemical Journal, (15 Dec 1998) 336/3 (711-717).
Refs: 50
ISSN: 0264-6021 CODEN: BIJOAK
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Control of oestradiol-responsive gene regulation by oestrogen receptors (ERs) may involve complex cross-talk with retinoic acid receptors (RARs)

and **retinoid** X receptors (RXRs). Recently, we have shown that ERα directly interacts with RARα and RXRα through their **ligand binding domains** (LBDs). In the present work, we extend these results by showing that ERβ **binds** similarly to RARα and RXRα but not to the glucocorticoid receptor, as demonstrated by the yeast **two-hybrid** tests and glutathione S-transferase pull-down assays. These direct interactions were also demonstrated in gel-shift assays, in which the oestrogen response element (ERE) **binding** by ERα was enhanced by the RXRα LED but was abolished by the RARα LED. In addition, we showed that RARα and RXRα. bound the ERE as efficiently as ERα, suggesting that competition for DNA **binding** may affect the **transactivation** function of the ER. In transient transfection experiments, co-expression of RARα or RXRα, along with ERα or ERβ, revealed differential modulation of the ERE-dependent **transactivation**, which was distinct from the results when each receptor alone was co-transfected. Importantly, when the LED of RARα was co-expressed with ERα, **transactivation** of ERα on the ERE was repressed as efficiently as when wild-type RARα was co-expressed. Furthermore, liganded RARα or unliganded RXRα enhanced the ERα **transactivation**, suggesting the formation of transcriptionally active heterodimer complexes between the ER and **retinoid** receptors. Taken together, these results suggest that direct protein-protein interactions may play major roles in the determination of the biological. . .

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ACCESSION NUMBER: 1998070270 EMBASE
TITLE: L7 protein is a coregulator of vitamin D receptor-retinoid X receptor- mediated transactivation.
AUTHOR: Berghofer-Hochheimer Y.; Zurek C.; Wolfl S.; Hemmerich P.; Munder T.
CORPORATE SOURCE: T. Munder, Hans-Knoll-Ins. Naturstoff-Forschung, Dept. of Cell and Molecular Biology, Beutenbergstr. 11, 07745 Jena, Germany
SOURCE: Journal of Cellular Biochemistry, (1 Apr 1998) 69/1 (1-12). Refs: 44
ISSN: 0730-2312 CODEN: JCEBD5
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The vitamin D receptor (VDR) heterodimerizes with the **retinoid** X receptor (RXR) and requires additional protein-protein interactions to regulate the expression of target genes. Using the yeast **two-hybrid** system, we identified the previously described protein L7, that specifically interacted with the VDR in the presence of vitamin D. Deletion analysis indicated, that the N-terminus of L7, which harbours a basic region leucine zipper like **domain**, mediated interaction with the VDR. **Binding** assays with purified GST-L7 demonstrated, that L7 specifically pulled down the VDR, that was either expressed in yeast or endogenously contained in the cell line U937. Interestingly, L7 inhibited **ligand**-dependent VDR-RXR heterodimerization, when constitutively expressed in yeast. We also demonstrate that L7 repressed **binding** of VDR-RXR heterodimers to a vitamin D response element. Surprisingly, L7 recruited RXR to the same response element in the presence of 9-cis retinoic acid. **Ligand**-dependent protein-protein interaction in the yeast **two-hybrid** system confirmed, that **binding** of L7 also was targeted at the RXR. Our data suggest, that protein L7 is a coregulator of VDR-RXR

mediated **transactivation** of genes, that modulates transcriptional activity by interfering with **binding** of the receptors to genomic enhancer elements.

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ACCESSION NUMBER: 96191450 EMBASE
DOCUMENT NUMBER: 1996191450
TITLE: An orphan nuclear hormone receptor that lacks a DNA binding domain and heterodimerizes with other receptors.
AUTHOR: Seol W.; Choi H.-S.; Moore D.D.
CORPORATE SOURCE: Department of Molecular Biology, Massachusetts General Hospital,Boston, MA 02114, United States
SOURCE: Science, (1996) 272/5266 (1336-1339).
ISSN: 0036-8075 CODEN: SCIEAS
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
AB SHP is an orphan member of the nuclear hormone receptor superfamily that contains the dimerization and **ligand-binding domain** found in other family members but lacks the conserved DNA **binding domain**. In the yeast **two-hybrid** system, SHP interacted with several conventional and orphan members of the receptor superfamily, including **retinoid** receptors, the thyroid hormone receptor, and the orphan receptor MB67. SHP also interacted directly with these receptors in vitro. In mammalian cells, SHP specifically inhibited **transactivation** by the superfamily members with which it interacted. These results suggest that SHP functions as a negative,regulator of receptor-dependent signaling. . .

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ACCESSION NUMBER: 95310518 EMBASE
DOCUMENT NUMBER: 1995310518
TITLE: A nuclear hormone receptor-associated protein that inhibits transactivation by the thyroid hormone and retinoic acid receptors.
AUTHOR: Burris T.P.; Nawaz Z.; Tsai M.-J.; O'Malley B.W.
CORPORATE SOURCE: Department of Cell Biology, Baylor College of Medicine, One Baylor Plaza,Houston, TX 77030, United States
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1995) 92/21 (9525-9529).
ISSN: 0027-8424 CODEN: PNASA6
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
AB . . . receptors are transcription factors that require multiple protein-protein interactions to regulate the expression of their target genes. Using the yeast **two-hybrid** system, we identified a protein, thyroid hormone receptor uncoupling protein (TRUP), that specifically interacts with a region of the human thyroid hormone receptor (TR) consisting of the hinge region and the N-terminal portion of the **ligand binding domain** in a hormone-independent manner. Interestingly, TRUP inhibits **transactivation** by TR and the retinoic acid receptor but has no effect on the estrogen receptor or the **retinoid** X receptor in mammalian cells. We also demonstrate that TRUP exerts its action on TR and retinoic acid receptor by. . .

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ACCESSION NUMBER: 94286001 EMBASE
DOCUMENT NUMBER: 1994286001
TITLE: [Paradoxical effect of retinoic acid in acute promyelocytic leukaemia].
LEUCEMIE AIGUE PROMYELOCYTAIRE ET ACIDE RETINOIQUE: LE PARADOXE.
AUTHOR: Lavau C.; Jansen J.; Weis K.; Lamond A.; Dejean A.
CORPORATE SOURCE: Un. Recomb./Expression Genetique, Inserm U.163, Institut Pasteur, 28, Rue du Docteur-Roux,75742 Paris Cedex 15, France
SOURCE: Medecine/Sciences, (1994) 10/8-9 (817-824).
ISSN: 0767-0974 CODEN: MSMSE4
COUNTRY: France
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 016 Cancer
029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index
LANGUAGE: French
SUMMARY LANGUAGE: French; English
AB . . . and γ) as members of the nuclear receptor superfamily led to important insights into the molecular mechanism of action of **retinoids**. The nuclear receptors, including also receptors for steroid hormone, vitamine D3 and thyroid hormone, act as **ligand** -inducible transcription factors and are characterized by the presensce of **two** well conserved DNA- and hormone-**binding** **domains**. One of the most intriguing properties of RA is its ability to induce in vivo differentiation of acute promyelocytic leukaemia. . . with APL, fuses an as yet unidentified gene, named PML, to the retinoic acid receptor α locus. The resulting PML-RARα **hybrid** protein that retains most of the functional **domains** of parental proteins exhibits altered **transactivating** functions when compared to the wild-type receptor; however, the biological significance of this property in the transforming phenotype is still. . . a novel family of nuclear proteins characterized by the presence of a Cys/His-rich motif, named a RING finger, that includes RNA-**binding** proteins, transcription factors and oncoproteins. A dimerization **domain** within PML is able to mediate the formation of PML-RARα homodimers that can **bind** to target sequences with distinct DNA **binding** properties if compared with RARα. Immunofluorescence studies have shown that PML is specifically localized within a discrete subnuclear compartment corresponding. . . to nuclear bodies recognized by patient autoimmune sera. These structures are distinct from snRNP-containing organelles. In APL cells, the PML-RARα **hybrid** that accumulates into abnormal substructures is able to delocalize the natural RARα partner, RXR, as well as some of the. . .

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ACCESSION NUMBER: 94322898 EMBASE
DOCUMENT NUMBER: 1994322898
TITLE: The peroxisome proliferator-activated receptor interacts with the retinoid X receptor in vivo.
AUTHOR: Miyata K.S.; McCaw S.E.; Marcus S.L.; Rachubinski R.A.; Capone J.P.
CORPORATE SOURCE: Department of Anatomy/Cell Biology, University of Alberta, Medical Sciences Building,Edmonton, Alta. T6G 2H7, Canada
SOURCE: Gene, (1994) 148/2 (327-330).
ISSN: 0378-1119 CODEN: GENED6
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 004 Microbiology
022 Human Genetics
029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The peroxisome proliferator-activated receptor (PPAR) **binds** cooperatively to cognate peroxisome proliferator-responsive elements (PPRE) in vitro through heterodimerization with **retinoid** X receptors (RXR). We used the yeast **two-hybrid** system to determine whether these **two** nuclear receptors physically interact in vivo. Mouse (m) PPAR and human (h) RXRα were synthesized as fusion proteins to either the DNA-**binding domain** (GBD) or the **transactivation domain** (GAD) of the yeast GAL4 transcription-activator protein, and were tested for their ability to activate expression of a GAL1::lacZ reporter. . . for the interaction of PPAR and RXRα in vivo in the absence of a PPRE target site or exogenously added **ligands**.